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Search  
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LV Cook 10/14/03

(FILE 'HOME' ENTERED AT 14:03:28 ON 14 OCT 2003)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT' ENTERED AT 14:03:52 ON  
14 OCT 2003

L1 2933 S IGA AND IGA1  
L2 1 S L1 AND SIDS?  
L3 347 S L1 AND ALTE?  
L4 195 DUPLICATE REMOVE L3 (152 DUPLICATES REMOVED)  
L5 12016 S (SUDDEN INFANT DEATH SYNDROME)  
L6 389 S (APPARENT LIFE THREATENING EVENTS)  
L7 18 S (ACUTE LIFE THREATENING EPISODES)  
L8 0 S L7 AND L1  
L9 1 S L6 AND L1  
L10 1 S L5 AND L1  
L11 69 S L1 AND INFLAMMATION?  
L12 1 S L2 AND INFECTION?  
L13 0 S L11 AND INFANT?  
L14 101 S L1 AND INFANT  
L15 1 S L14 AND DEATH?  
L16 3321 S (UPPER RESPIRATORY INFECTION)  
L17 0 S L16 AND L14  
L18 6 S L16 AND L1  
L19 3 DUPLICATE REMOVE L18 (3 DUPLICATES REMOVED)  
L20 129 S L5 AND IG?  
L21 49 S L5 AND IGA?  
L22 21 DUPLICATE REMOVE L21 (28 DUPLICATES REMOVED)  
L23 1 S L6 AND IGA?

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L22 21 DUPLICATE REMOVE L21 (28 DUPLICATES REMOVED)

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22 ANSWER 3 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 2  
 AN 1999:291661 BIOSIS  
 DN PREV199900291661  
 TI IL-6 cerebrospinal fluid levels are related to laryngeal **IgA** and  
 epithelial HLA-DR response in **sudden infant  
 death syndrome**.  
 AU Vege, Ashild (1); Rognum, Torleiv Ole; Anestad, Gabriel  
 CS (1) Institute of Forensic Medicine, National Hospital, N-0027, Oslo Norway  
 SO Pediatric Research, (June, 1999) Vol. 45, No. 6, pp. 803-809.  
 ISSN: 0031-3998.  
 DT Article ↳ check date ✓  
 LA English  
 SL English  
 AB The objective was to investigate whether there is any correlation between  
 signs of central and peripheral immune stimulation in victims of  
**sudden infant death syndrome** (SIDS),  
 the former expressed by IL-6 in cerebrospinal fluid (CSF), the latter by  
**IgA**, IgG, and IgM immunocytes, T lymphocytes, and HLA-DR  
 expression in laryngeal mucosa. Seventeen SIDS cases with low CSF IL-6  
 levels (ltoreq5 pg/mL) and 20 cases with high CSF IL-6 levels (gtoreq30  
 pg/mL) were subjected to immunohistochemical quantitation of **IgA**  
 , IgG, and IgM immunocytes; semiquantitative scoring of T lymphocytes in  
 the mucosa of epiglottis and larynx, and semiquantitative evaluation of  
 HLA-DR expression. SIDS cases with IL-6 levels gtoreq30 pg/mL had a  
 significantly higher number of **IgA** immunocytes in laryngeal  
 mucosa (p = 0.007) and in epiglottis (p = 0.03) than cases with IL-6  
 levels ltoreq5 pg/mL. Furthermore, laryngeal HLA-DR expression was  
 significantly more extensive in SIDS cases with IL-6 levels gtoreq30 pg/mL  
 than in those with levels ltoreq5 pg/mL (p = 0.05). No differences were  
 found for IgG and IgM immunocytes or for T cells. In addition, babies  
 found prone more often had symptoms of slight infection before death and  
 had a higher number of **IgA** immunocytes in the larynx (p = 0.02)  
 than babies sleeping on their side or back. Because IL-6 levels gtoreq30  
 pg/mL correspond to the levels found in infants who die from infectious  
 diseases such as meningitis/septicemia and pneumonia, the findings favor  
 the hypothesis that many SIDS cases may be caused by an "overreaction" of  
 the immune system to an otherwise harmless infection.  
 CC Immunology and Immunochemistry - General; Methods \*34502  
 Cytology and Cytochemistry - Human \*02508  
 Biochemical Studies - General \*10060  
 Biophysics - General Biophysical Studies \*10502  
 Respiratory System - General; Methods \*16001  
 Nervous System - General; Methods \*20501  
 Pediatrics \*25000  
 BC Hominidae 86215  
 IT Major Concepts  
 Clinical Immunology (Human Medicine, Medical Sciences); Neurology  
 (Human Medicine, Medical Sciences); Pediatrics (Human Medicine, Medical  
 Sciences); Pulmonary Medicine (Human Medicine, Medical Sciences)  
 IT Parts, Structures, & Systems of Organisms  
 cerebrospinal fluid: nervous system; epiglottis: mucosa, respiratory  
 system; immunocytes: immune system; larynx: mucosa, respiratory system;  
 T lymphocytes: blood and lymphatics, immune system  
 IT Diseases  
**sudden infant death syndrome**:  
 disease-miscellaneous  
 IT Chemicals & Biochemicals  
 HLA-DR: expression; **IgA** [immunoglobulin A]; IgG  
 [immunoglobulin G]; IgM [immunoglobulin M]; IL-6 [interleukin-6]:  
 cerebrospinal fluid  
 IT Alternate Indexing

Sudden Infant Death (MeSH)

ORGN Super Taxa

Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name

human (Hominidae): female, infant, male

ORGN Organism Superterms

Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L22 ANSWER 4 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 3  
 AN 1999:355526 BIOSIS  
 DN PREV199900355526  
 TI The protective effect of breast feeding in relation to **sudden infant death syndrome** (SIDS): III. Detection of **IgA** antibodies in human milk that bind to bacterial toxins implicated in SIDS.  
 AU Gordon, Ann E. (1); Saadi, Abdulrahman T.; MacKenzie, Doris A. C.; Molony, Neil; James, Valerie S.; Weir, Donald M.; Busuttil, Anthony; Blackwell, C. Caroline  
 CS (1) Department of Medical Microbiology, Medical School, University of Edinburgh, Teviot Place, Edinburgh, EH8 9AG UK  
 SO FEMS Immunology and Medical Microbiology, (Aug. 1, 1999) Vol. 25, No. 1-2, pp. 175-182.  
 ISSN: 0928-8244.  
 DT Article  
 LA English  
 SL English  
 AB Two toxin-producing bacteria implicated in **sudden infant death syndrome** (SIDS) are *Staphylococcus aureus* and *Clostridium perfringens*. Epidemiological studies have shown that breast feeding reduces an infant's risk of SIDS. This protective effect could be due partly to **IgA** antibodies to these toxins in human milk. The aim of this work was to use a quantitative ELISA to determine levels of **IgA** antibodies that bound to toxic shock syndrome toxin (TSST-1), staphylococcal enterotoxin C (SEC) and *C. perfringens* enterotoxin A (CEA) in individual samples of human milk. All samples of milk tested contained **IgA** antibodies that bound to the bacterial toxins. For individual samples, **IgA** bound to TSST-1, SEC and CEA were in the range of 900-3100 ng ml<sup>-1</sup>, 1000-3600 ng ml<sup>-1</sup> and 1000-4300 ng ml<sup>-1</sup> respectively. Isolation of *S. aureus* from mothers donating breast milk samples was used to determine if the presence of bacteria affected **IgA** levels which bound TSST-1 and SEC. For 3/5 samples with levels above the upper limit of the standard deviation (2375 ng ml<sup>-1</sup>) for **IgA** bound to TSST-1, *S. aureus* was isolated from the mother whilst 4/5 samples found to contain levels above the upper limit of the standard deviation (2627 ng ml<sup>-1</sup>) for **IgA** bound to SEC, had *S. aureus* isolated from the mother. In conclusion, if bacterial toxins do play a role in precipitating a SIDS death, the presence of **IgA** antibodies to toxins in breast milk, but not in infant formula, might contribute to the protective effect of breast feeding in relation to SIDS.  
 CC Biochemical Studies - General \*10060  
 Respiratory System - General; Methods \*16001  
 Toxicology - General; Methods and Experimental \*22501  
 Bacteriology, General and Systematic \*30000  
 Medical and Clinical Microbiology - General; Methods and Techniques \*36001  
 BC Micrococcaceae 07702  
 Endospore-forming Gram-Positives 07810  
 Hominidae 86215  
 IT Major Concepts  
 Biochemistry and Molecular Biophysics; Toxicology  
 IT Diseases  
**sudden infant death syndrome:**  
 disease-miscellaneous  
 IT Chemicals & Biochemicals  
 toxic shock syndrome toxin; **IgA** [immunoglobulin A]; Lewis a antigen; Lewis b antigen  
 IT Alternate Indexing  
 Sudden Infant Death (MeSH)  
 IT Methods & Equipment

date  
no good

flow cytometry: analytical method

IT Miscellaneous Descriptors  
breast feeding; human milk; infant formula preparation

ORGN Super Taxa  
Endospore-forming Gram-Positives: Eubacteria, Bacteria, Microorganisms;  
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia;  
Micrococcaceae: Gram-Positive Cocci, Eubacteria, Bacteria,  
Microorganisms

ORGN Organism Name  
human (Hominidae): infant, patient; Clostridium perfringens  
(Endospore-forming Gram-Positives): binding, toxigenic; Staphylococcus  
aureus (Micrococcaceae)

ORGN Organism Superterms  
Animals; Bacteria; Chordates; Eubacteria; Humans; Mammals;  
Microorganisms; Primates; Vertebrates

22 ANSWER 3 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 2

AN 1999:291661 BIOSIS

DN PREV199900291661

TI IL-6 cerebrospinal fluid levels are related to laryngeal **IgA** and  
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AU Vege, Ashild (1); Rognum, Torleiv Ole; Anestad, Gabriel

CS (1) Institute of Forensic Medicine, National Hospital, N-0027, Oslo Norway

SO Pediatric Research, (June, 1999) Vol. 45, No. 6, pp. 803-809.  
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DT Article

LA English

SL English

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 Cytology and Cytochemistry - Human \*02508  
 Biochemical Studies - General \*10060  
 Biophysics - General Biophysical Studies \*10502  
 Respiratory System - General; Methods \*16001  
 Nervous System - General; Methods \*20501  
 Pediatrics \*25000

BC Hominidae 86215

IT Major Concepts  
 Clinical Immunology (Human Medicine, Medical Sciences); Neurology  
 (Human Medicine, Medical Sciences); Pediatrics (Human Medicine, Medical  
 Sciences); Pulmonary Medicine (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms  
 cerebrospinal fluid: nervous system; epiglottis: mucosa, respiratory  
 system; immunocytes: immune system; larynx: mucosa, respiratory system;  
 T lymphocytes: blood and lymphatics, immune system

IT Diseases  
**sudden infant death syndrome:**  
 disease-miscellaneous

IT Chemicals & Biochemicals  
 HLA-DR: expression; **IgA** [immunoglobulin A]; IgG  
 [immunoglobulin G]; IgM [immunoglobulin M]; IL-6 [interleukin-6]:  
 cerebrospinal fluid

IT Alternate Indexing

Sudden Infant Death (MeSH)

ORGN Super Taxa

Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name

human (Hominidae): female, infant, male

ORGN Organism Superterms

Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L22 ANSWER 4 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
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 AU Gordon, Ann E. (1); Saadi, Abdulrahman T.; MacKenzie, Doris A. C.; Molony,  
 Neil; James, Valerie S.; Weir, Donald M.; Busuttil, Anthony; Blackwell, C.  
 Caroline  
 CS (1) Department of Medical Microbiology, Medical School, University of  
 Edinburgh, Teviot Place, Edinburgh, EH8 9AG UK  
 SO FEMS Immunology and Medical Microbiology, (Aug. 1, 1999) Vol. 25, No. 1-2,  
 pp. 175-182.  
 ISSN: 0928-8244. *date no good*  
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 of breast feeding in relation to SIDS.  
 CC Biochemical Studies - General \*10060  
 Respiratory System - General; Methods \*16001  
 Toxicology - General; Methods and Experimental \*22501  
 Bacteriology, General and Systematic \*30000  
 Medical and Clinical Microbiology - General; Methods and Techniques  
 \*36001  
 BC Micrococcaceae 07702  
 Endospore-forming Gram-Positives 07810  
 Hominidae 86215  
 IT Major Concepts  
 Biochemistry and Molecular Biophysics; Toxicology  
 IT Diseases  
**sudden infant death syndrome:**  
 disease-miscellaneous  
 IT Chemicals & Biochemicals  
 toxic shock syndrome toxin; **IgA** [immunoglobulin A]; Lewis a  
 antigen; Lewis b antigen  
 IT Alternate Indexing  
 Sudden Infant Death (MeSH)  
 IT Methods & Equipment

flow cytometry: analytical method

IT Miscellaneous Descriptors  
breast feeding; human milk; infant formula preparation

ORGN Super Taxa  
Endospore-forming Gram-Positives: Eubacteria, Bacteria, Microorganisms;  
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia;  
Micrococcaceae: Gram-Positive Cocci, Eubacteria, Bacteria,  
Microorganisms

ORGN Organism Name  
human (Hominidae): infant, patient; Clostridium perfringens  
(Endospore-forming Gram-Positives): binding, toxigenic; Staphylococcus  
aureus (Micrococcaceae)

ORGN Organism Superterms  
Animals; Bacteria; Chordates; Eubacteria; Humans; Mammals;  
Microorganisms; Primates; Vertebrates

L22 ANSWER 6 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 5  
 AN 1999:365762 BIOSIS  
 DN PREV199900365762  
 TI Immunological evidence for a bacterial toxin aetiology in **sudden  
 infant death syndrome**.  
 AU Siarakas, Steven (1); Brown, Alissa Jane; Murrell, William G.  
 CS (1) Department of Microbiology and Infectious Diseases, Concord  
 Repatriation General Hospital, Concord, NSW, 2139 Australia  
 SO FEMS Immunology and Medical Microbiology, (Aug. 1, 1999) Vol. 25, No. 1-2,  
 pp. 37-50.  
 ISSN: 0928-8244. *date no good*  
 DT Article  
 LA English  
 SL English  
 AB Toxin-specific antibodies to clostridial, enterobacterial and  
 staphylococcal toxins implicated in **sudden infant  
 death syndrome** were studied in sera from **sudden  
 infant death syndrome** infants and a comparison  
 group of infants (babies with phenylketonuria). The results indicated a  
 higher proportion of sera from **sudden infant  
 death syndrome** infants contained **IgA** that  
 bound to clostridial and enterobacterial toxins but a higher proportion of  
 sera from the phenylketonuria comparison group contained **IgA**  
 that bound staphylococcal toxins. The higher proportion of serum samples  
 with IgG and IgM in the healthy comparison babies serum probably indicated  
 immunity in this group of babies to these toxins. The effect of gender and  
 age had a minimal effect on the incidence of these antibodies. The  
 presence of toxin-specific antibodies in **sudden infant  
 death syndrome** and the of comparison infants suggests  
 that all infants are exposed to these toxins and most babies successfully  
 overcome the toxic challenge. Some infants with predisposing risk factors  
 (temperature change, smoking, infection, immune development, sleeping  
 position, etc.) that could affect the baby's immune competency could  
 succumb to these and possibly other toxins. This immunological evidence  
 further strengthens the view that bacterial toxins are a significant cause  
 of **sudden infant death syndrome**.  
 CC Toxicology - General; Methods and Experimental \*22501  
 Pathology, General and Miscellaneous - Diagnostic \*12504  
 Pediatrics \*25000  
 Medical and Clinical Microbiology - General; Methods and Techniques  
 \*36001  
 Pathology, General and Miscellaneous - Therapy \*12512  
 BC Hominidae 86215  
 IT Major Concepts  
 Pediatrics (Human Medicine, Medical Sciences); Toxicology  
 IT Diseases  
**sudden infant death syndrome:**  
 disease-miscellaneous  
 IT Chemicals & Biochemicals  
 clostridial toxins; enterobacterial toxins; staphylococcal toxins;  
**IgA** [immunoglobulin A]; IgG [immunoglobulin G]; IgM  
 [immunoglobulin M]  
 IT Alternate Indexing  
 Sudden Infant Death (MeSH)  
 ORGN Super Taxa  
 Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia  
 ORGN Organism Name  
 human (Hominidae): infant, patient  
 ORGN Organism Superterms  
 Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L22 ANSWER 6 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 5

AN 1999:365762 BIOSIS

DN PREV199900365762

TI Immunological evidence for a bacterial toxin aetiology in **sudden infant death syndrome**.

AU Siarakas, Steven (1); Brown, Alissa Jane; Murrell, William G.

CS (1) Department of Microbiology and Infectious Diseases, Concord Repatriation General Hospital, Concord, NSW, 2139 Australia

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DT Article

LA English

SL English

AB Toxin-specific antibodies to clostridial, enterobacterial and staphylococcal toxins implicated in **sudden infant death syndrome** were studied in sera from **sudden infant death syndrome** infants and a comparison group of infants (babies with phenylketonuria). The results indicated a higher proportion of sera from **sudden infant death syndrome** infants contained **IgA** that bound to clostridial and enterobacterial toxins but a higher proportion of sera from the phenylketonuria comparison group contained **IgA** that bound staphylococcal toxins. The higher proportion of serum samples with IgG and IgM in the healthy comparison babies serum probably indicated immunity in this group of babies to these toxins. The effect of gender and age had a minimal effect on the incidence of these antibodies. The presence of toxin-specific antibodies in **sudden infant death syndrome** and the of comparison infants suggests that all infants are exposed to these toxins and most babies successfully overcome the toxic challenge. Some infants with predisposing risk factors (temperature change, smoking, infection, immune development, sleeping position, etc.) that could affect the baby's immune competency could succumb to these and possibly other toxins. This immunological evidence further strengthens the view that bacterial toxins are a significant cause of **sudden infant death syndrome**.

CC Toxicology - General; Methods and Experimental \*22501  
 Pathology, General and Miscellaneous - Diagnostic \*12504  
 Pediatrics \*25000  
 Medical and Clinical Microbiology - General; Methods and Techniques \*36001  
 Pathology, General and Miscellaneous - Therapy \*12512

BC Hominidae 86215

IT Major Concepts  
 Pediatrics (Human Medicine, Medical Sciences); Toxicology

IT Diseases  
**sudden infant death syndrome:**  
 disease-miscellaneous

IT Chemicals & Biochemicals  
 clostridial toxins; enterobacterial toxins; staphylococcal toxins;  
**IgA** [immunoglobulin A]; IgG [immunoglobulin G]; IgM [immunoglobulin M]

IT Alternate Indexing  
 Sudden Infant Death (MeSH)

ORGN Super Taxa  
 Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name  
 human (Hominidae): infant, patient

ORGN Organism Superterms  
 Animals; Chordates; Humans; Mammals; Primates; Vertebrates

date no  
good

L22 ANSWER 8 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 7

AN 1996:69320 BIOSIS

DN PREV199698641455

TI Pulmonary eosinophilia in **sudden infant death syndrome**.

AU Baxendine, Julia A.; Moore, Isabella E. (1)

CS (1) Dep. Histopathol., Level E, Southampton General Hosp., Southampton SO9 4XY UK

SO Journal of Pathology, (1995) Vol. 177, No. 4, pp. 415-421. *ejournal*  
 ISSN: 0022-3417.

DT Article

LA English

AB A recent immunohistochemical study found increased numbers of eosinophils, but no mast cells, in the pulmonary parenchyma of infants who died of **sudden infant death syndrome (SIDS)**.  
 The present study tested the hypothesis that this pulmonary eosinophilia could be IgE-mediated. Histomorphometry was used to compare the numbers of eosinophils, mast cells, and IgG-, **IgA**-, IgM- and IgE-expressing lymphoid cells in the lungs of two groups of infants. Twenty-eight subjects aged less than 1 year were selected from postmortem records of infant deaths between 1989 and 1992. Fourteen were cases of SIDS and these infants were matched for age and gender to 14 controls who died of other non-pulmonary conditions. Immunohistochemical stains were used and positive cells were counted on six peribronchial and six subpleural fields. The numbers of eosinophils in both peribronchial and subpleural regions were significantly higher in SIDS compared with controls (P=0.0071 and P=0.041, respectively). The numbers of **IgA**-expressing lymphoid cells were also significantly increased in SIDS cases (P=0.042). There were no differences in IgG, IgM or IgE expression or in mast cell numbers. These results confirmed that pulmonary eosinophils are increased in SIDS, but not through an IgE-mediated pathway.

CC Microscopy Techniques - Histology and Histochemistry \*01056  
 Cytology and Cytochemistry - Human 02508  
 Biochemistry - Gases \*10012  
 Biochemical Studies - General 10060  
 Biochemical Studies - Proteins, Peptides and Amino Acids 10064  
 Biochemical Studies - Carbohydrates 10068  
 Anatomy and Histology, General and Comparative - Microscopic and Ultramicroscopic Anatomy \*11108  
 Pathology, General and Miscellaneous - Necrosis \*12510  
 Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies \*15004  
 Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System \*15008  
 Respiratory System - Pathology \*16006  
 Pediatrics \*25000  
 Immunology and Immunochemistry - Immunopathology, Tissue Immunology \*34508

BC Hominidae \*86215

IT Major Concepts  
 Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Methods and Techniques; Morphology; Pathology; Pediatrics (Human Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine, Medical Sciences)

IT Miscellaneous Descriptors  
 AIRWAY OBSTRUCTION; HYPOXIA; IMMUNOGLOBULIN A; IMMUNOGLOBULIN E; IMMUNOGLOBULIN G; IMMUNOGLOBULIN M; PEDIATRICS

ORGN Super Taxa  
 Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name  
 Hominidae (Hominidae)

ORGN Organism Superterms

animals; chordates; humans; mammals; primates; vertebrates

L22 ANSWER 9 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN DUPLICATE 8

AN 95119681 EMBASE  
DN 1995119681

TI Changes in the concentration and distribution of immunoglobulin-producing  
cells in SIDS palatine tonsils.

AU Stoltenberg L.; Vege A.; Saugstad O.D.; Rognum T.O.  
CS Department of Pediatric Research, National Hospital, N-0027 Oslo, Norway  
SO Pediatric Allergy and Immunology, (1995) 6/1 (48-55).  
ISSN: 0905-6157 CODEN: PALUEE

CY Denmark  
DT Journal; Article  
FS 007 Pediatrics and Pediatric Surgery  
026 Immunology, Serology and Transplantation

LA English  
SL English  
AB Seventeen **sudden infant death syndrome** (SIDS) cases and 9 controls, were examined immunohistochemically with regard to the presence of **IgA**-, **IgM**-, **IgD**, and **IgG**, as well as for the subtypes **IgG1**-, **IgG2**-, **IgC3**-, and **IgG4**-immunocytes. Differences in compartmentalization were also investigated. Differences were demonstrated between SIDS and controls in total number of **IgG** cells per 0.1 mm<sup>2</sup> tissue area (median: 18.3, range: 12.3-30.2 versus median: 6.3, range: 2.0-14.6) ( $p < 0.01$ ), and for **IgA** immunocytes (median: 3.9, range: 2.4-5.0 versus median: 1.5, range: 1.1-3.7) ( $p < 0.05$ ), while no differences were demonstrated for **IgM** cells (median: 1.8, range: 1.2-3.3 versus median: 1.8, range: 0.7-5.6) or **IgD** cells (median: 1.9, range: 0.8-2.9 versus median: 1.6, range: 0.7-2.4). Differences were demonstrated between SIDS and control **IgG** plasma cells in all the four palatine tonsillar compartments; germinal centre ( $p < 0.01$ ), mantle zone ( $p < 0.05$ ), interfollicular area ( $p < 0.01$ ) and reticular epithelium ( $p < 0.01$ ). Furthermore, the number of **IgA** cells was higher in SIDS vs. controls in both the germinal centre (median: 1.4, range: 0.6-2.1 versus median: 0.6, range: 0.3-1.3) ( $p < 0.05$ ) and in the interfollicular area (median: 2.2, range: 1.1-3.1 versus median: 0.5, range: 0.4-2.0) ( $p < 0.01$ ). For **IgM** immunocytes, differences were demonstrated in the germinal centre (median: 1.0, range: 0.4-1.6 versus median: 0.4, range: 0.3-1.3) ( $p < 0.01$ ) as well as in the germinal centre (median: 0.6, range: 0.5-0.8 versus median: 0.4, range: 0.3-0.7) ( $p < 0.01$ ) and in the interfollicular area (median: 1.2, range: 0.8-1.6 versus median: 0.5, range: 0.5-0.7) ( $p < 0.01$ ) in the **IgD** immunocyte group. The total number of **IgG1**- and **IgG3**-immunocytes were increased in SIDS (median 15.6, range: 5.3-58.9 versus median: 2.5, range: 1.5-8.4) ( $p < 0.01$ ) and (median: 3, 6, range: 0.4-8.6 versus median: 1.1, range: 0.4-1.3) ( $p < 0.01$ ) respectively. Furthermore, significantly increased numbers of these two subclasses, were seen in all the four compartments in the SIDS cases. The palatine tonsillar immune system is stimulated in SIDS. Furthermore, the changes being predominantly in the germinal centres and interfollicular areas, are indicating a recent stimulation, and the **IgG**-subgroup response pattern makes a viral protein antigen the most likely stimulant.

CT Medical Descriptors:  
\*immunoglobulin producing cell  
\*palatine tonsil  
\***sudden infant death syndrome**  
article  
clinical article  
controlled study  
female  
germinal center  
human  
human cell

human tissue  
immunohistochemistry  
infant  
lymphocyte  
male

priority journal

Drug Descriptors:

immunoglobulin a: EC, endogenous compound  
immunoglobulin d: EC, endogenous compound  
immunoglobulin g1: EC, endogenous compound  
immunoglobulin g2: EC, endogenous compound  
immunoglobulin g3: EC, endogenous compound  
immunoglobulin g4: EC, endogenous compound  
immunoglobulin m: EC, endogenous compound  
virus antigen  
virus protein

RN (immunoglobulin m) 9007-85-6

L22 ANSWER 10 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 9

AN 1993:343465 BIOSIS

DN PREV199396040465

TI Mucosal immune response in a case of **sudden infant death syndrome**.

AU Gleeson, Maree (1); Clancy, Robert L.; Cripps, Allan W.

CS (1) Hunter Immunology Unit, Royal Newcastle Hosp., PO Box 664J, Newcastle  
 NSW 2300 Australia

SO Pediatric Research, (1993) Vol. 33, No. 6, pp. 554-556.  
 ISSN: 0031-3998.

DT Article

LA English

AB A prospective study to define the normal patterns of development of  
 mucosal immunity in 263 children provided a unique opportunity to study  
 the mucosal immune response in an infant who unexpectedly died from  
**sudden infant death syndrome**. The  
 subject initially had a normal pattern of mucosal immune function, which  
 was perturbed after a transient mild upper respiratory tract infection at  
 3 1/2 wk of age. After the upper respiratory tract infection, there was an  
 increase in mucosal permeability and the appearance of **IgA** and  
 IgM in saliva. The unusual features in this case were the degree and the  
 duration of the increases in salivary **IgA** and IgM after  
 resolution of the illness. The marked abnormalities suggested a persistent  
 stimulation of the mucosal immune response. The case provides informative  
 data on potential mechanisms of **sudden infant death syndrome** and supports a role for involvement of  
 upper respiratory tract infection.

CC Biochemical Studies - Proteins, Peptides and Amino Acids \*10064  
 Biochemical Studies - Carbohydrates \*10068  
 Pathology, General and Miscellaneous - Necrosis \*12510  
 Respiratory System - Pathology \*16006  
 Pediatrics \*25000  
 Immunology and Immunochemistry - Bacterial, Viral and Fungal \*34504  
 Medical and Clinical Microbiology - General; Methods and Techniques  
 \*36001

BC Hominidae \*86215

IT Major Concepts  
 Biochemistry and Molecular Biophysics; Immune System (Chemical  
 Coordination and Homeostasis); Infection; Pathology; Pediatrics (Human  
 Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine,  
 Medical Sciences)

IT Industry  
 cattle industry

IT Miscellaneous Descriptors  
 IMMUNOGLOBULIN M TITERS; VACCINES

ORGN Super Taxa  
 Bacteria - General Unspecified: Eubacteria, Bacteria; Bovidae:  
 Artiodactyla, Mammalia, Vertebrata, Chordata, Animalia;  
 Enterobacteriaceae: Eubacteria, Bacteria; Hominidae: Primates,  
 Mammalia, Vertebrata, Chordata, Animalia; Mammalia - Unspecified:  
 Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name  
 bacteria (Bacteria - General Unspecified); cow (Bovidae); mammal  
 (Mammalia - Unspecified); microorganism (Microorganisms - Unspecified);  
 Escherichia coli (Enterobacteriaceae); Hominidae (Hominidae)

ORGN Organism Superterms  
 animals; artiodactyls; bacteria; chordates; eubacteria; humans;  
 mammals; microorganisms; nonhuman mammals; nonhuman vertebrates;  
 primates; vertebrates

L22 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2003 ACS on STN  
 AN 2000:881419 CAPLUS  
 DN 134:16545  
 TI Determining susceptibility for acute life-threatening events and/or SIDS  
 IN Clancy, Robert; Gleeson, Maree  
 PA University of Newcastle Research Associates Limited, Australia  
 SO PCT Int. Appl., 32 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 IC ICM G01N033-53  
 ICS G01N033-68  
 CC 15-3 (Immunochemistry)  
 Section cross-reference(s): 14  
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000075656	A1	20001214	WO 2000-AU643	20000607
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1185866	A1	20020313	EP 2000-930888	20000607
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	BR 2000011424	A	20020326	BR 2000-11424	20000607
	JP 2003501659	T2	20030114	JP 2001-501880	20000607
	NO 2001005923	A	20020207	NO 2001-5923	20011204
	ZA 2002000081	A	20021211	ZA 2002-81	20020104
PRAI	AU 1999-810	A	19990607		
	WO 2000-AU643	W	20000607		

AB The authors disclose that predisposition to apparent life-threatening events (ALTE) and/or **sudden infant death syndrome** (SIDS) is assocd. with elevated total salivary **IgA** and/or **IgA1** subsequent to upper respiratory tract infection.

ST susceptibility SIDS apnea **IgA**

IT Immunoglobulins  
 RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (A, secretory; as marker for apparent life-threatening events and/or SIDS in humans)

IT Immunoglobulins  
 RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (A1; as marker for apparent life-threatening events and/or SIDS in humans)

IT Immunoglobulins  
 RL: ANT (Analyte); ANST (Analytical study)  
 (G; susceptibility for apparent life-threatening events and/or SIDS in relation to elevated levels of **IgA** in comparison to)

IT Apnea  
 Biomarkers (biological responses)  
 Risk assessment  
 (**IgA** as marker for apparent life-threatening events and/or SIDS in humans)

IT Immunoglobulins

RL: ANT (Analyte); ANST (Analytical study)  
 (M; susceptibility for apparent life-threatening events and/or SIDS in relation to elevated levels of **IgA** in comparison to)

IT Proteins, specific or class  
 RL: ANT (Analyte); ANST (Analytical study)  
 (acute-phase; susceptibility for apparent life-threatening events and/or SIDS in relation to elevated levels of **IgA** in comparison to)

IT Immunoassay  
 (enzyme-linked immunosorbent assay; for detn. of salivary **IgA** and susceptibility to apparent life-threatening events and/or SIDS)

IT Immunoassay  
 (immunodiffusion; for detn. of salivary **IgA** and susceptibility to apparent life-threatening events and/or SIDS)

IT Immunoglobulins  
 RL: ANT (Analyte); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)  
 (secretory; assocn. with susceptibility for apparent life-threatening events and/or SIDS)

IT Death  
 (**sudden infant death syndrome**;  
**IgA** as marker for susceptibility to)

IT Body fluid  
 Saliva  
 (susceptibility for apparent life-threatening events and/or SIDS in relation to elevated levels of **IgA** in)

IT Respiratory tract  
 (upper, infection; **IgA** as marker for apparent life-threatening events and/or SIDS in humans assocd. with)

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

- (1) Beach, P; US 5556759 A 1996 CAPLUS
- (2) Beach, P; US 5747266 A 1998 CAPLUS
- (3) Gleeson, M; Pediatric Research 1993, V33(6), P554 MEDLINE

L22 ANSWER 2 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 1

AN 2000:297990 BIOSIS

DN PREV200000297990

TI Immunohistochemical examination of the lungs in infant death cases using antibodies against milk components.

AU Iwadate, Kimiharu (1); Doy, Mikio; Nishimaki, Yuko; Liang, Fang; Takatori, Takehiko; Hasekura, Hayato

CS (1) Department of Legal Medicine, Faculty of Medicine, Tokyo Medical and Dental University, Yushima 1-5-45, Bunkyo-ku, Tokyo, 113-8519 Japan

SO Forensic Science International, (May 8th, 2000) Vol. 110, No. 1, pp. 19-28. print.

ISSN: 0379-0738.

DT Article

LA English

SL English

AB To examine the use of immunohistochemical staining with antibodies against milk components for detection of aspirated milk on lung sections, eighteen infant death cases were investigated. Immunostaining was performed with anti-human alpha lactalbumin, anti-human **IgA**, anti-human milk fat globulin 1, and anti-cow whey antibody. Reactivity with each antibody was examined, and semi-quantitative examinations were performed to compare the amount of aspirated milk using anti-human alpha lactalbumin antibody. Materials in the alveoli or bronchioli on lung sections suspected to be aspirated milk showed the most sensitive and clearest reaction with anti-human alpha lactalbumin antibody. Of the eighteen cases, ten cases showed positive reaction with this antibody. The amount of aspirated milk

varied widely in each case. In conclusion, immunohistochemical staining with antibodies against human milk components, especially anti-human alpha lactalbumin antibody, can detect small amounts of milk. Using this method, we were able to compare the relative amount of aspirated milk among cases.

CC General Biology - Forensic Science . \*00531  
 Biochemical Studies - General \*10060  
 Biophysics - General Biophysical Studies \*10502  
 Pathology, General and Miscellaneous - General \*12502  
 Pediatrics \*25000  
 Immunology and Immunochemistry - General; Methods \*34502

BC Hominidae 86215

IT Major Concepts  
 Biochemistry and Molecular Biophysics; Immune System (Chemical Coordination and Homeostasis); Forensics

IT Parts, Structures, & Systems of Organisms  
 lung secretions: aspirated milk, respiratory system

IT Diseases  
**sudden infant death syndrome:**  
 disease-miscellaneous

IT Chemicals & Biochemicals  
 anti-cow whey antibody; anti-human **IgA** antibody [anti-human immunoglobulin A antibody]; anti-human alpha-lactalbumin antibody; anti-human milk fat globulin 1 antibody

IT Alternate Indexing  
 Sudden Infant Death (MeSH)

IT Methods & Equipment  
 immunohistochemical staining: staining method

ORGN Super Taxa  
 Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name  
 human (Hominidae)

ORGN Organism Superterms  
 Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L22 ANSWER 3 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 2

AN 1999:291661 BIOSIS

DN PREV199900291661

TI IL-6 cerebrospinal fluid levels are related to laryngeal **IgA** and epithelial HLA-DR response in **sudden infant death syndrome**.

AU Vege, Ashild (1); Rognum, Torleiv Ole; Anestad, Gabriel

CS (1) Institute of Forensic Medicine, National Hospital, N-0027, Oslo Norway

SO Pediatric Research, (June, 1999) Vol. 45, No. 6, pp. 803-809.  
 ISSN: 0031-3998.

DT Article

LA English

SL English

AB The objective was to investigate whether there is any correlation between signs of central and peripheral immune stimulation in victims of **sudden infant death syndrome** (SIDS), the former expressed by IL-6 in cerebrospinal fluid (CSF), the latter by **IgA**, IgG, and IgM immunocytes, T lymphocytes, and HLA-DR expression in laryngeal mucosa. Seventeen SIDS cases with low CSF IL-6 levels (ltoreq5 pg/mL) and 20 cases with high CSF IL-6 levels (gtoreq30 pg/mL) were subjected to immunohistochemical quantitation of **IgA**, IgG, and IgM immunocytes; semiquantitative scoring of T lymphocytes in the mucosa of epiglottis and larynx, and semiquantitative evaluation of HLA-DR expression. SIDS cases with IL-6 levels gtoreq30 pg/mL had a significantly higher number of **IgA** immunocytes in laryngeal mucosa (p = 0.007) and in epiglottis (p = 0.03) than cases with IL-6 levels ltoreq5 pg/mL. Furthermore, laryngeal HLA-DR expression was

significantly more extensive in SIDS cases with IL-6 levels  $\geq 30$  pg/mL than in those with levels  $\leq 5$  pg/mL ( $p = 0.05$ ). No differences were found for IgG and IgM immunocytes or for T cells. In addition, babies found prone more often had symptoms of slight infection before death and had a higher number of **IgA** immunocytes in the larynx ( $p = 0.02$ ) than babies sleeping on their side or back. Because IL-6 levels  $\geq 30$  pg/mL correspond to the levels found in infants who die from infectious diseases such as meningitis/septicemia and pneumonia, the findings favor the hypothesis that many SIDS cases may be caused by an "overreaction" of the immune system to an otherwise harmless infection.

CC Immunology and Immunochemistry - General; Methods \*34502  
 Cytology and Cytochemistry - Human \*02508  
 Biochemical Studies - General \*10060  
 Biophysics - General Biophysical Studies \*10502  
 Respiratory System - General; Methods \*16001  
 Nervous System - General; Methods \*20501  
 Pediatrics \*25000

BC Hominidae 86215

IT Major Concepts  
 Clinical Immunology (Human Medicine, Medical Sciences); Neurology (Human Medicine, Medical Sciences); Pediatrics (Human Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms  
 cerebrospinal fluid: nervous system; epiglottis: mucosa, respiratory system; immunocytes: immune system; larynx: mucosa, respiratory system; T lymphocytes: blood and lymphatics, immune system

IT Diseases  
**sudden infant death syndrome:**  
 disease-miscellaneous

IT Chemicals & Biochemicals  
 HLA-DR: expression; **IgA** [immunoglobulin A]; IgG [immunoglobulin G]; IgM [immunoglobulin M]; IL-6 [interleukin-6]: cerebrospinal fluid

IT Alternate Indexing  
 Sudden Infant Death (MeSH)

ORGN Super Taxa  
 Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name  
 human (Hominidae): female, infant, male

ORGN Organism Superterms  
 Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L22 ANSWER 4 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 3

AN 1999:355526 BIOSIS

DN PREV199900355526

TI The protective effect of breast feeding in relation to **sudden infant death syndrome** (SIDS): III. Detection of **IgA** antibodies in human milk that bind to bacterial toxins implicated in SIDS.

AU Gordon, Ann E. (1); Saadi, Abdulrahman T.; MacKenzie, Doris A. C.; Molony, Neil; James, Valerie S.; Weir, Donald M.; Busuttill, Anthony; Blackwell, C. Caroline

CS (1) Department of Medical Microbiology, Medical School, University of Edinburgh, Teviot Place, Edinburgh, EH8 9AG UK

SO FEMS Immunology and Medical Microbiology, (Aug. 1, 1999) Vol. 25, No. 1-2, pp. 175-182.  
 ISSN: 0928-8244.

DT Article

LA English

SL English

AB Two toxin-producing bacteria implicated in **sudden infant**

**death syndrome** (SIDS) are *Staphylococcus aureus* and *Clostridium perfringens*. Epidemiological studies have shown that breast feeding reduces an infant's risk of SIDS. This protective effect could be due partly to **IgA** antibodies to these toxins in human milk. The aim of this work was to use a quantitative ELISA to determine levels of **IgA** antibodies that bound to toxic shock syndrome toxin (TSST-1), staphylococcal enterotoxin C (SEC) and *C. perfringens* enterotoxin A (CEA) in individual samples of human milk. All samples of milk tested contained **IgA** antibodies that bound to the bacterial toxins. For individual samples, **IgA** bound to TSST-1, SEC and CEA were in the range of 900-3100 ng ml<sup>-1</sup>, 1000-3600 ng ml<sup>-1</sup> and 1000-4300 ng ml<sup>-1</sup> respectively. Isolation of *S. aureus* from mothers donating breast milk samples was used to determine if the presence of bacteria affected **IgA** levels which bound TSST-1 and SEC. For 3/5 samples with levels above the upper limit of the standard deviation (2375 ng ml<sup>-1</sup>) for **IgA** bound to TSST-1, *S. aureus* was isolated from the mother whilst 4/5 samples found to contain levels above the upper limit of the standard deviation (2627 ng ml<sup>-1</sup>) for **IgA** bound to SEC, had *S. aureus* isolated from the mother. In conclusion, if bacterial toxins do play a role in precipitating a SIDS death, the presence of **IgA** antibodies to toxins in breast milk, but not in infant formula, might contribute to the protective effect of breast feeding in relation to SIDS.

- CC Biochemical Studies - General \*10060
  - Respiratory System - General; Methods \*16001
  - Toxicology - General; Methods and Experimental \*22501
  - Bacteriology, General and Systematic \*30000
  - Medical and Clinical Microbiology - General; Methods and Techniques \*36001
- BC Micrococcaceae 07702
  - Endospore-forming Gram-Positives 07810
  - Hominidae 86215
- IT Major Concepts
  - Biochemistry and Molecular Biophysics; Toxicology
- IT Diseases
  - sudden infant death syndrome:**
  - disease-miscellaneous
- IT Chemicals & Biochemicals
  - toxic shock syndrome toxin; **IgA** [immunoglobulin A]; Lewis a antigen; Lewis b antigen
- IT Alternate Indexing
  - Sudden Infant Death (MeSH)
- IT Methods & Equipment
  - flow cytometry: analytical method
- IT Miscellaneous Descriptors
  - breast feeding; human milk; infant formula preparation
- ORGN Super Taxa
  - Endospore-forming Gram-Positives: Eubacteria, Bacteria, Microorganisms;
  - Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia;
  - Micrococcaceae: Gram-Positive Cocci, Eubacteria, Bacteria, Microorganisms
- ORGN Organism Name
  - human (Hominidae): infant, patient; *Clostridium perfringens* (Endospore-forming Gram-Positives): binding, toxigenic; *Staphylococcus aureus* (Micrococcaceae)
- ORGN Organism Superterms
  - Animals; Bacteria; Chordates; Eubacteria; Humans; Mammals; Microorganisms; Primates; Vertebrates
- L22 ANSWER 5 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 4
- AN 1999:355524 BIOSIS
- DN PREV199900355524

TI The protective effect of breast feeding in relation to **sudden infant death syndrome** (SIDS): I. The effect of human milk and infant formula preparations on binding of toxigenic *Staphylococcus aureus* to epithelial cells.

AU Saadi, Abdulrahman T.; Gordon, Ann E.; MacKenzie, Doris A. C.; James, Valerie S.; Elton, Robert A.; Weir, Donald M.; Busuttil, Anthony; Blackwell, C. Caroline (1)

CS (1) Department of Medical Microbiology, Medical School, University of Edinburgh, Teviot Place, Edinburgh, EH8 9AG UK

SO FEMS Immunology and Medical Microbiology, (Aug. 1, 1999) Vol. 25, No. 1-2, pp. 155-165.  
ISSN: 0928-8244.

DT Article

LA English

SL English

AB Epidemiological studies indicate that breast-fed infants are at a decreased risk of **sudden infant death syndrome** (SIDS) compared to formula-fed infants. Increasing evidence suggests that infectious agents might be involved in some of these deaths, in particular bacteria which colonise mucosal surfaces and produce superantigenic toxins. One species implicated in recent studies of SIDS infants is *Staphylococcus aureus*. We tested the hypothesis that in comparison to infant formula, human milk might be a better inhibitor of binding of *S. aureus* to epithelial cells. In this study, two protocols were used for the binding assays which were assessed by flow cytometry: the in vitro method in which bacteria were treated with milk or formula, washed and added to epithelial cells; and a method more closely reflecting the competitive interactions in vivo in which cells, bacteria, and milk or infant formula were added at the same time. With the in vivo method, breast milk caused enhancement of bacterial binding to cells whilst infant formula caused inhibition; however, for the in vitro method, both human milk and infant formula caused consistent enhancement of binding. Flow cytometry and light microscopy studies indicated that the enhancement was due to the formation of bacterial aggregates. Human milk and infant formula preparations were also compared for components (antibodies or oligosaccharides) that could inhibit binding of *S. aureus* using the in vitro method. Human milk contained both **IgA** and IgG. Neither human milk nor infant formula contained oligosaccharides reactive with the *Ulex europaeus* lectin but both contained components that bound monoclonal antibodies to Lewis<sup>a</sup> and Lewis<sup>b</sup> antigens which can act as receptors for *S. aureus*. With both methods, synthetic Lewis<sup>a</sup> and Lewis<sup>b</sup> inhibited *S. aureus* binding in a dose-dependent manner. With human milk, however, the only component which showed a significant correlation with inhibition of binding was the **IgA** specific for the staphylococcal surface component that binds Lewis<sup>a</sup>. Both human milk and infant formula contain components which could potentially inhibit bacterial binding but only breast milk contains the **IgA** specific for the bacterial adhesin that binds Lewis<sup>a</sup>. Studies using the in vivo method suggest that protection associated with breast feeding in relation to SIDS could be due mainly to the formation of bacterial aggregates. The studies have implications for further research into constituents of infant formula.

CC Biochemical Studies - General \*10060  
Respiratory System - General; Methods \*16001  
Toxicology - General; Methods and Experimental \*22501  
Bacteriology, General and Systematic \*30000  
Medical and Clinical Microbiology - General; Methods and Techniques \*36001

BC Micrococcaceae 07702  
Hominidae 86215

IT Major Concepts  
Biochemistry and Molecular Biophysics; Toxicology

IT Diseases

**sudden infant death syndrome:**  
disease-miscellaneous

IT Chemicals & Biochemicals  
**IgA** [immunoglobulin A]; IgG [immunoglobulin G]

IT Alternate Indexing  
Sudden Infant Death (MeSH)

IT Methods & Equipment  
flow cytometry: analytical method

IT Miscellaneous Descriptors  
human milk; infant formula preparation

ORGN Super Taxa  
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia;  
Micrococcaceae: Gram-Positive Cocci, Eubacteria, Bacteria,  
Microorganisms

ORGN Organism Name  
human (Hominidae): infant, patient; Staphylococcus aureus  
(Micrococcaceae): binding, toxigenic

ORGN Organism Superterms  
Animals; Bacteria; Chordates; Eubacteria; Humans; Mammals;  
Microorganisms; Primates; Vertebrates

L22 ANSWER 6 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 5

AN 1999:365762 BIOSIS

DN PREV199900365762

TI Immunological evidence for a bacterial toxin aetiology in **sudden  
infant death syndrome.**

AU Siarakas, Steven (1); Brown, Alissa Jane; Murrell, William G.

CS (1) Department of Microbiology and Infectious Diseases, Concord  
Repatriation General Hospital, Concord, NSW, 2139 Australia

SO FEMS Immunology and Medical Microbiology, (Aug. 1, 1999) Vol. 25, No. 1-2,  
pp. 37-50.  
ISSN: 0928-8244.

DT Article

LA English

SL English

AB Toxin-specific antibodies to clostridial, enterobacterial and  
staphylococcal toxins implicated in **sudden infant  
death syndrome** were studied in sera from **sudden  
infant death syndrome** infants and a comparison  
group of infants (babies with phenylketonuria). The results indicated a  
higher proportion of sera from **sudden infant  
death syndrome** infants contained **IgA** that  
bound to clostridial and enterobacterial toxins but a higher proportion of  
sera from the phenylketonuria comparison group contained **IgA**  
that bound staphylococcal toxins. The higher proportion of serum samples  
with IgG and IgM in the healthy comparison babies serum probably indicated  
immunity in this group of babies to these toxins. The effect of gender and  
age had a minimal effect on the incidence of these antibodies. The  
presence of toxin-specific antibodies in **sudden infant  
death syndrome** and the of comparison infants suggests  
that all infants are exposed to these toxins and most babies successfully  
overcome the toxic challenge. Some infants with predisposing risk factors  
(temperature change, smoking, infection, immune development, sleeping  
position, etc.) that could affect the baby's immune competency could  
succumb to these and possibly other toxins. This immunological evidence  
further strengthens the view that bacterial toxins are a significant cause  
of **sudden infant death syndrome.**

CC Toxicology - General; Methods and Experimental \*22501  
Pathology, General and Miscellaneous - Diagnostic \*12504  
Pediatrics \*25000  
Medical and Clinical Microbiology - General; Methods and Techniques

\*36001  
 Pathology, General and Miscellaneous - Therapy \*12512  
 BC Hominidae 86215  
 IT Major Concepts  
     Pediatrics (Human Medicine, Medical Sciences); Toxicology  
 IT Diseases  
     **sudden infant death syndrome:**  
     disease-miscellaneous  
 IT Chemicals & Biochemicals  
     clostridial toxins; enterobacterial toxins; staphylococcal toxins;  
     **IgA** [immunoglobulin A]; IgG [immunoglobulin G]; IgM  
     [immunoglobulin M]  
 IT Alternate Indexing  
     Sudden Infant Death (MeSH)  
 ORGN Super Taxa  
     Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia  
 ORGN Organism Name  
     human (Hominidae): infant, patient  
 ORGN Organism Superterms  
     Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L22 ANSWER 7 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.  
 on STN DUPLICATE 6

AN 1998218426 EMBASE  
 TI Maturation of B cells in the lamina propria of human gut and bronchi in  
 the first months of human life.  
 AU El Kaissouni J.; Bene M.C.; Thionnois S.; Monin P.; Vidailhet M.; Faure  
 G.C.  
 CS G.C. Faure, Laboratoire d'Immunologie, Faculte de Medicine, BP 184,  
 Vandoeuvre les Nancy 54500, France  
 SO Developmental Immunology, (1998) 5/3 (153-159).  
 Refs: 27  
 ISSN: 1044-6672 CODEN: DEIME7  
 CY United Kingdom  
 DT Journal; Article  
 FS 021 Developmental Biology and Teratology  
     026 Immunology, Serology and Transplantation  
 LA English  
 SL English  
 AB Little is known of the maturation of the mucosae-associated lymphoid  
 tissue (MALT) in man, because, for ethical reasons, tissues from newborns  
 are not easy to obtain. We used the opportunity provided by autopsies  
 systematically performed in infants who died of **Sudden**  
**Infant Death Syndrome** (SIDS) to study the  
 maturation of the MALT after birth. Gut and bronchus samples of 90 infants  
 from postpartum to 90 months and who died from SIDS were collected and  
 studied by histological and immunofluorescence examination. Plasma cells,  
 absent at birth, appeared within a few hours after birth and initially  
 were of the IgM isotype. **IgA** plasma cells appeared at 12 days.  
 These cells were first observed in gut and later in bronchi, indicating  
 that maturation of the gut precedes that of bronchi. The number of plasma  
 cells increased rapidly over time and **IgA** plasma cells became  
 predominant after 3 weeks in the gut and 6 weeks in bronchi. At birth,  
 only small IgM bearing B-cell foci were seen and organized germinal  
 centers appeared to develop over a few days, first in the gut and only  
 later in bronchi. These results confirm that, in man, the MALT  
 organization at birth is still in its fetal form and that maturation  
 depends on intestinal challenges and evolves over several weeks before  
**IgA** becomes the predominant isotype secreted.

CT Medical Descriptors:  
 \*b lymphocyte  
 \*cell maturation

\*lamina propria  
intestine  
bronchus  
lymphoid tissue

**sudden infant death syndrome**

autopsy  
immunofluorescence test  
histology  
plasma cell  
germinal center  
human  
male  
female  
human tissue  
infant  
preschool child

article

priority journal

Drug Descriptors:

immunoglobulin m: EC, endogenous compound

immunoglobulin a: EC, endogenous compound

RN (immunoglobulin m) 9007-85-6

L22 ANSWER 8 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 7

AN 1996:69320 BIOSIS

DN PREV199698641455

TI Pulmonary eosinophilia in **sudden infant death syndrome**.

AU Baxendine, Julia A.; Moore, Isabella E. (1)

CS (1) Dep. Histopathol., Level E, Southampton General Hosp., Southampton SO9  
4XY UK

SO Journal of Pathology, (1995) Vol. 177, No. 4, pp. 415-421.  
ISSN: 0022-3417.

DT Article

LA English

AB A recent immunohistochemical study found increased numbers of eosinophils,  
but no mast cells, in the pulmonary parenchyma of infants who died of  
**sudden infant death syndrome** (SIDS).

The present study tested the hypothesis that this pulmonary eosinophilia  
could be IgE-mediated. Histomorphometry was used to compare the numbers of  
eosinophils, mast cells, and IgG-, **IgA**-, IgM- and IgE-expressing  
lymphoid cells in the lungs of two groups of infants. Twenty-eight  
subjects aged less than 1 year were selected from postmortem records of  
infant deaths between 1989 and 1992. Fourteen were cases of SIDS and these  
infants were matched for age and gender to 14 controls who died of other  
non-pulmonary conditions. Immunohistochemical stains were used and  
positive cells were counted on six peribronchial and six subpleural  
fields. The numbers of eosinophils in both peribronchial and subpleural  
regions were significantly higher in SIDS compared with controls (P=0.0071  
and P=0.041, respectively). The numbers of **IgA**-expressing  
lymphoid cells were also significantly increased in SIDS cases (P=0.042).  
There were no differences in IgG, IgM or IgE expression or in mast cell  
numbers. These results confirmed that pulmonary eosinophils are increased  
in SIDS, but not through an IgE-mediated pathway.

CC Microscopy Techniques - Histology and Histochemistry \*01056

Cytology and Cytochemistry - Human 02508

Biochemistry - Gases \*10012

Biochemical Studies - General 10060

Biochemical Studies - Proteins, Peptides and Amino Acids 10064

Biochemical Studies - Carbohydrates 10068

Anatomy and Histology, General and Comparative - Microscopic and

Ultramicroscopic Anatomy \*11108  
Pathology, General and Miscellaneous - Necrosis \*12510  
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies \*15004  
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and  
Reticuloendothelial System \*15008  
Respiratory System - Pathology \*16006  
Pediatrics \*25000  
Immunology and Immunochemistry - Immunopathology, Tissue Immunology  
\*34508

BC Hominidae \*86215

IT Major Concepts

Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport  
and Circulation); Clinical Immunology (Human Medicine, Medical  
Sciences); Methods and Techniques; Morphology; Pathology; Pediatrics  
(Human Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine,  
Medical Sciences)

IT Miscellaneous Descriptors

AIRWAY OBSTRUCTION; HYPOXIA; IMMUNOGLOBULIN A; IMMUNOGLOBULIN E;  
IMMUNOGLOBULIN G; IMMUNOGLOBULIN M; PEDIATRICS

ORGN Super Taxa

Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name

Hominidae (Hominidae)

ORGN Organism Superterms

animals; chordates; humans; mammals; primates; vertebrates

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on STN DUPLICATE 8

AN 95119681 EMBASE

DN 1995119681

TI Changes in the concentration and distribution of immunoglobulin-producing  
cells in SIDS palatine tonsils.

AU Stoltenberg L.; Vege A.; Saugstad O.D.; Rognum T.O.

CS Department of Pediatric Research, National Hospital, N-0027 Oslo, Norway

SO Pediatric Allergy and Immunology, (1995) 6/1 (48-55).

ISSN: 0905-6157 CODEN: PALUEE

CY Denmark

DT Journal; Article

FS 007 Pediatrics and Pediatric Surgery

026 Immunology, Serology and Transplantation

LA English

SL English

AB Seventeen **sudden infant death**

**syndrome** (SIDS) cases and 9 controls, were examined  
immunohistochemically with regard to the presence of **IgA**-, **IgM**-,  
**IgD**, and **IgG**, as well as for the subtypes **IgG1**-, **IgG2**-, **IgC3**-, and  
**IgG4**-immunocytes. Differences in compartmentalization were also  
investigated. Differences were demonstrated between SIDS and controls in  
total number of **IgG** cells per 0.1 mm<sup>2</sup> tissue area (median: 18.3, range:  
12.3-30.2 versus median: 6.3, range: 2.0-14.6) ( $p < 0.01$ ), and for  
**IgA** immunocytes (median: 3.9, range: 2.4-5.0 versus median: 1.5,  
range: 1.1-3.7) ( $p < 0.05$ ), while no differences were demonstrated for **IgM**  
cells (median: 1.8, range: 1.2-3.3 versus median: 1.8, range: 0.7-5.6) or  
**IgD** cells (median: 1.9, range: 0.8-2.9 versus median: 1.6, range:  
0.7-2.4). Differences were demonstrated between SIDS and control **IgG**  
plasma cells in all the four palatine tonsillar compartments; germinal  
centre ( $p < 0.01$ ), mantle zone ( $p < 0.05$ ), interfollicular area ( $p < 0.01$ )  
and reticular epithelium ( $p < 0.01$ ). Furthermore, the number of  
**IgA** cells was higher in SIDS vs. controls in both the germinal  
centre (median: 1.4, range: 0.6-2.1 versus median: 0.6, range: 0.3-1.3) ( $p$   
 $< 0.05$ ) and in the interfollicular area (median: 2.2, range: 1.1-3.1  
versus median: 0.5, range: 0.4-2.0) ( $p < 0.01$ ). For **IgM** immunocytes,

differences were demonstrated in the germinal centre (median: 1.0, range: 0.4-1.6 versus median: 0.4, range: 0.3-1.3) ( $p < 0.01$ ) as well as in the germinal centre (median: 0.6, range: 0.5-0.8 versus median: 0.4, range: 0.3-0.7) ( $p < 0.01$ ) and in the interfollicular area (median: 1.2, range: 0.8-1.6 versus median: 0.5, range: 0.5-0.7) ( $p < 0.01$ ) in the IgD immunocyte group. The total number of IgG1- and IgG3-immunocytes were increased in SIDS (median 15.6, range: 5.3-58.9 versus median: 2.5, range: 1.5-8.4) ( $p < 0.01$ ) and (median: 3, 6, range: 0.4-8.6 versus median: 1.1, range: 0.4-1.3) ( $p < 0.01$ ) respectively. Furthermore, significantly increased numbers of these two subclasses, were seen in all the four compartments in the SIDS cases. The palatine tonsillar immune system is stimulated in SIDS. Furthermore, the changes being predominantly in the germinal centres and interfollicular areas, are indicating a recent stimulation, and the IgG-subgroup response pattern makes a viral protein antigen the most likely stimulant.

CT Medical Descriptors:

\*immunoglobulin producing cell  
 \*palatine tonsil  
 \*sudden infant death syndrome

article  
 clinical article  
 controlled study  
 female  
 germinal center  
 human  
 human cell  
 human tissue  
 immunohistochemistry  
 infant  
 lymphocyte  
 male

priority journal

Drug Descriptors:

immunoglobulin a: EC, endogenous compound  
 immunoglobulin d: EC, endogenous compound  
 immunoglobulin g1: EC, endogenous compound  
 immunoglobulin g2: EC, endogenous compound  
 immunoglobulin g3: EC, endogenous compound  
 immunoglobulin g4: EC, endogenous compound  
 immunoglobulin m: EC, endogenous compound  
 virus antigen  
 virus protein

RN (immunoglobulin m) 9007-85-6

L22 ANSWER 10 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 9

AN 1993:343465 BIOSIS

DN PREV199396040465

TI Mucosal immune response in a case of **sudden infant death syndrome**.

AU Gleeson, Maree (1); Clancy, Robert L.; Cripps, Allan W.

CS (1) Hunter Immunology Unit, Royal Newcastle Hosp., PO Box 664J, Newcastle  
 NSW 2300 Australia

SO Pediatric Research, (1993) Vol. 33, No. 6, pp. 554-556..  
 ISSN: 0031-3998.

DT Article

LA English

AB A prospective study to define the normal patterns of development of mucosal immunity in 263 children provided a unique opportunity to study the mucosal immune response in an infant who unexpectedly died from **sudden infant death syndrome**. The subject initially had a normal pattern of mucosal immune function, which

was perturbed after a transient mild upper respiratory tract infection at 3 1/2 wk of age. After the upper respiratory tract infection, there was an increase in mucosal permeability and the appearance of **IgA** and **IgM** in saliva. The unusual features in this case were the degree and the duration of the increases in salivary **IgA** and **IgM** after resolution of the illness. The marked abnormalities suggested a persistent stimulation of the mucosal immune response. The case provides informative data on potential mechanisms of **sudden infant death syndrome** and supports a role for involvement of upper respiratory tract infection.

CC Biochemical Studies - Proteins, Peptides and Amino Acids \*10064  
 Biochemical Studies - Carbohydrates \*10068  
 Pathology, General and Miscellaneous - Necrosis \*12510  
 Respiratory System - Pathology \*16006  
 Pediatrics \*25000  
 Immunology and Immunochemistry - Bacterial, Viral and Fungal \*34504  
 Medical and Clinical Microbiology - General; Methods and Techniques \*36001

BC Hominidae \*86215

IT Major Concepts  
 Biochemistry and Molecular Biophysics; Immune System (Chemical Coordination and Homeostasis); Infection; Pathology; Pediatrics (Human Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine, Medical Sciences)

IT Industry  
 cattle industry

IT Miscellaneous Descriptors  
 IMMUNOGLOBULIN M TITERS; VACCINES

ORGN Super Taxa  
 Bacteria - General Unspecified: Eubacteria, Bacteria; Bovidae: Artiodactyla, Mammalia, Vertebrata, Chordata, Animalia; Enterobacteriaceae: Eubacteria, Bacteria; Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia; Mammalia - Unspecified: Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name  
 bacteria (Bacteria - General Unspecified); cow (Bovidae); mammal (Mammalia - Unspecified); microorganism (Microorganisms - Unspecified); Escherichia coli (Enterobacteriaceae); Hominidae (Hominidae)

ORGN Organism Superterms  
 animals; artiodactyls; bacteria; chordates; eubacteria; humans; mammals; microorganisms; nonhuman mammals; nonhuman vertebrates; primates; vertebrates

L22 ANSWER 11 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 10

AN 1992:258253 BIOSIS

DN BA93:134578

TI **SUDDEN INFANT DEATH SYNDROME**  
 VICTIMS SHOW LOCAL IMMUNOGLOBULIN M RESPONSE IN TRACHEAL WALL AND IMMUNOGLOBULIN A RESPONSE IN DUODENAL MUCOSA.

AU STOLTENBERG L; SAUGSTAD O D; ROGNUM T O

CS INST. FORENSIC MED., UNIV. OSLO, NATIONAL HOSP., N-0027 OSLO, NORWAY.

SO PEDIATR RES, (1992) 31 (4 PART 1), 372-375.  
 CODEN: PEREBL. ISSN: 0031-3998.

FS BA; OLD

LA English

AB Twenty-two **sudden infant death syndrome** (SIDS) cases and 22 controls were examined immunohistochemically with regard to **IgA**, **IgM**, and **IgG** plasma cells in tracheal wall and duodenal mucosa. Furthermore, the presence of secretory component in tracheal surface and gland epithelium as well as in duodenal crypt and villus epithelium were evaluated. The examined

specimens were obtained at autopsies. The control groups consisted of 11 infants who died of noninfectious causes and 11 who died of infections. In the tracheal wall, the SIDS group had higher IgM cell numbers than the control group that died of noninfectious causes ( $p < 0.01$ ), whereas the SIDS victims had lower **IgA** and IgM cell numbers than the infectious control group ( $p < 0.01$ ). In the duodenal mucosa, the SIDS group had significantly higher **IgA** cell numbers than the noninfectious control group ( $p < 0.02$ ) but lower **IgA** cell numbers than the infection group ( $p < 0.01$ ). Secretory component was present in the epithelium from all SIDS cases and controls, both in the tracheal wall glands and in the duodenal crypt mucosa. These findings indicate that the mucosal immune system is stimulated in SIDS.

CC Mathematical Biology and Statistical Methods 04500  
 Biochemical Studies - Proteins, Peptides and Amino Acids 10064  
 Biochemical Studies - Carbohydrates 10068  
 Biophysics - Membrane Phenomena \*10508  
 Pathology, General and Miscellaneous - Necrosis \*12510  
 Digestive System - Physiology and Biochemistry \*14004  
 Respiratory System - Physiology and Biochemistry \*16004  
 Pediatrics \*25000  
 Immunology and Immunochemistry - Immunopathology, Tissue Immunology \*34508

BC Hominidae 86215

IT Miscellaneous Descriptors

#### HUMAN IMMUNOGLOBULIN G MUCOSAL IMMUNE SYSTEM STATISTICS

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 on STN

AN 91285019 EMBASE

DN 1991285019

TI Ontogeny of the mucosal immune system and **IgA** deficiency.

AU Brandtzaeg P.; Nilssen D.E.; Rognum T.O.; Thrane P.S.

CS LIIPAT, Institutt for Patologi, Rikshospitalet, N-0027 Oslo 1, Norway

SO Gastroenterology Clinics of North America, (1991) 20/3 (397-439).

ISSN: 0889-8553 CODEN: GCNAEF

CY United States

DT Journal; General Review

FS 007 Pediatrics and Pediatric Surgery

026 Immunology, Serology and Transplantation

048 Gastroenterology

LA English

SL English

AB Studies of ontogenesis contribute to better understanding of regulatory events underlying the striking heterogeneity in B-cell differentiation pathways employed in the human mucosal immune system. This disparity is probably explained by exogenous environmental factors, although regional differences probably also exist in accessory cells and cytokines involved in local immune responses. **IgA** deficiency signifies a heterogeneous syndrome but is basically a manifestation of a defect in B-cell differentiation. The immaturity of the **IgA** system revealed in this disorder bears a striking resemblance to that seen in newborn infants. It may therefore be worthwhile to consider **IgA** deficiency in relation to the ontogeny of mucosal immunity.

CT Medical Descriptors:

\*breast feeding

\*human immunodeficiency virus

\*immunity

\*ontogeny

\*sudden infant death syndrome: ET, etiology

child

female

human

human tissue  
male  
newborn  
nutrition  
review  
t lymphocyte  
Drug Descriptors:  
\*immunoglobulin a: EC, endogenous compound

L22 ANSWER 13 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
AN 1990:510848 BIOSIS  
DN BR39:122844  
TI SIDS VICTIMS SHOW LOCAL IGM RESPONSE IN TRACHEAL WALL AND **IGA**  
RESPONSE IN DUODENAL MUCOSA.  
AU STOLTENBERG L; SAUGSTAD O D; BRANDTZAEG P; ROGNUM T O  
CS FORENSIC DEP., CHILD. CLIN., NATL. HOSP., PILESTREDET 32, 0027 OSLO 1,  
NORWAY.  
SO 1990 ANNUAL MEETING OF THE EUROPEAN SOCIETY FOR PEDIATRIC RESEARCH,  
VIENNA, AUSTRIA, SEPTEMBER 23-27, 1990. PEDIATR RES. (1990) 28 (3), 277.  
CODEN: PEREBL. ISSN: 0031-3998.  
DT Conference  
FS BR; OLD  
LA English  
CC General Biology - Symposia, Transactions and Proceedings of Conferences,  
Congresses, Review Annuals 00520  
Cytology and Cytochemistry - Human \*02508  
Clinical Biochemistry; General Methods and Applications 10006  
Biochemical Studies - Proteins, Peptides and Amino Acids 10064  
Biochemical Studies - Carbohydrates 10068  
Pathology, General and Miscellaneous - Diagnostic 12504  
Pathology, General and Miscellaneous - Necrosis \*12510  
Metabolism - Carbohydrates \*13004  
Metabolism - Proteins, Peptides and Amino Acids \*13012  
Digestive System - Physiology and Biochemistry \*14004  
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies  
15002  
Respiratory System - Physiology and Biochemistry \*16004  
Pediatrics \*25000  
Immunology and Immunochemistry - General; Methods 34502  
Immunology and Immunochemistry - Immunopathology, Tissue Immunology  
\*34508  
BC Hominidae 86215  
IT Miscellaneous Descriptors  
ABSTRACT CHILD IMMUNOGLOBULIN M IMMUNOGLOBULIN A HLA IMMUNOLOGY  
**SUDDEN INFANT DEATH SYNDROME**

L22 ANSWER 14 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 11  
AN 1989:136492 BIOSIS  
DN BA87:71145  
TI LUNG IMMUNOGLOBULINS IN THE **SUDDEN INFANT**  
**DEATH SYNDROME.**  
AU FORSYTH K D; WEEKS S C; KOH L; SKINNER J; BRADLEY J  
CS DEP. IMMUNOL., INST. CHILD HEALTH, UNIV. LONDON, LONDON WC1N 1EH.  
SO BR MED J, (1989) 298 (6665), 23-26.  
CODEN: BMJOAE. ISSN: 0007-1447.  
FS BA; OLD  
LA English  
AB The incidence of the **sudden infant death**  
**syndrome** parallels that of respiratory tract infections in the  
paediatric community. On the basis that the aetiology of the  
**sudden infant death syndrome** may lie

in an unusual response to a trivial intercurrent respiratory infection a necropsy study was carried out investigating pulmonary immunoglobulins in 16 victims of the syndrome and a series of infants (controls) who had died of non-pulmonary causes. Compared with the controls victims of the **sudden infant death syndrome** had grossly raised concentrations of IgG, IgM, and to a less extent **IgA** in lung lavage samples. In addition, pulmonary interstitial and terminal airway cells expressing these immunoglobulins were identified far more often in victims than controls. The study failed to determine whether the increased immunoglobulin concentrations were a consequence of an unusual response to a trivial infection or an expression of otherwise altered immunological control in the respiratory tract. Epidemiological evidence and the findings of this study suggest that the respiratory tract is the prime target organ in the **sudden infant death syndrome**.

- CC Cytology and Cytochemistry - Human \*02508  
 Clinical Biochemistry; General Methods and Applications 10006  
 Biochemical Methods - Proteins, Peptides and Amino Acids 10054  
 Biochemical Studies - Proteins, Peptides and Amino Acids \*10064  
 Pathology, General and Miscellaneous - Necrosis \*12510  
 Respiratory System - Pathology \*16006  
 Pediatrics \*25000  
 Immunology and Immunochemistry - Immunopathology, Tissue Immunology \*34508  
 Medical and Clinical Microbiology - General; Methods and Techniques \*36001  
 Public Health: Epidemiology - Organic Diseases and Neoplasms \*37054
- BC Microorganisms - Unspecified 01000  
 Hominidae 86215
- IT Miscellaneous Descriptors  
 HUMAN RESPIRATORY TRACT INFECTIONS EPIDEMIOLOGICAL EVIDENCE  
 IMMUNOGLOBULIN M IMMUNOGLOBULIN G IMMUNOGLOBULIN A PULMONARY  
 INTERSTITIAL CELLS TERMINAL AIRWAY CELLS
- L22 ANSWER 15 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.  
 on STN
- AN 85080518 EMBASE  
 DN 1985080518  
 TI [Sudden death in children: Histological appearance of the salivary].  
 PLOTZLICHER KINDSTOD: HISTOLOGISCHE BEFUNDE IN DEN KOPFSPEICHELDRUSEN.  
 AU Molz G.; Hartmann H.P.; Michels L.  
 CS Anatomisches Institut, Universitat Zurich, CH-8057 Zurich, Switzerland  
 SO Pathologie, (1985) 6/1 (8-12).  
 CODEN: PATHDE
- CY Germany  
 DT Journal
- FS 005 General Pathology and Pathological Anatomy  
 007 Pediatrics and Pediatric Surgery  
 011 Otorhinolaryngology  
 049 Forensic Science Abstracts
- LA German
- AB Histologic research on parotid and submandibular salivary glands of 25 victims of sudden infant death, revealed in 17 cases (14%) pathologic changes. Cytomegalovirus infection was diagnosed in 13 cases and in 2 cases acute and in 2 cases chronic inflammations. Serologic examination of 7 cases revealed cytomegalovirus specific IgM in one and in 4 others positive IgG. One of the two infants with a chronic interstitial inflammation was also IgG positive. Among 275 infants of the same age group who died of clinical disease, 14 (5%) had cytomegalovirus. In the control group as in the test group cytomegalovirus was found more often in first born and migrant children. These results bear relevance for sudden infant death insofar as salivary glands are essential in the production

of secretory **IgA**.

CT Medical Descriptors:

\*cytomegalic inclusion body disease

\*salivary gland

\*sialoadenitis

**\*sudden infant death syndrome**

histology

etiology

autopsy

major clinical study

human

mouth

L22 ANSWER 16 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 12

AN 1979:265073 BIOSIS

DN BA68:67577

TI BOUND IMMUNO GLOBULIN AND FOREIGN ANTIGEN IN LUNGS OF **SUDDEN  
INFANT DEATH SYNDROME** VICTIMS.

AU ACKERMANN W W; EVELAND W C; MAVERAKIS N H; RAVEN C; GOLDEN A

CS DEP. EPIDEMIOLOG., UNIV. MICH., ANN ARBOR, MICH. 48109, USA.

SO INFECT IMMUN, (1979) 24 (3), 925-931.

CODEN: INFIBR. ISSN: 0019-9567.

FS BA; OLD

LA English

AB Lung sections from 33 infants who died suddenly and unexpectedly and who were diagnosed by medical examiners as **sudden infant death syndrome** (SIDS) gave evidence of bound immunoglobulin (Ig)G when examined by direct fluorescent antibody technique. Ten tissues from appropriate control infants were negative. Specimens containing IgG exhibited no **IgA** or IgE, but 3 contained IgM. Of lung sections with IgG, 61% contained .KAPPA. or .lambda. antigens. The remainder contained both. The indirect fluorescent antibody technique gave similar results. Blood sera of some individuals in the study which were tested all contained .KAPPA. and .lambda. antigens. Fluorescent-labeled Ig from 1 SIDS victim stained 7 of 17 SIDS lung sections tested, including his own. Labeled Ig from 3 mothers of SIDS victims exhibited differential selectivity in reaction with antigen in lungs of a group of 18 SIDS infants. They did not react with 10 control infant tissues. Various labeled adult sera, cord sera and serum from an apneic child did not react with the various lungs of SIDS victims in the study.

CC Biochemical Methods - Proteins, Peptides and Amino Acids 10054

Biochemical Methods - Carbohydrates 10058

Biochemical Studies - Proteins, Peptides and Amino Acids 10064

Biochemical Studies - Carbohydrates 10068

Pathology, General and Miscellaneous - Necrosis \*12510

Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies  
15002

Respiratory System - Physiology and Biochemistry \*16004

Respiratory System - Pathology \*16006

Pediatrics \*25000

Developmental Biology - Embryology - General and Descriptive 25502

Immunology and Immunochemistry - General; Methods 34502

Immunology and Immunochemistry - Immunopathology, Tissue Immunology  
\*34508

BC Hominidae 86215

IT Miscellaneous Descriptors

IMMUNO GLOBULIN G IMMUNO GLOBULIN M

L22 ANSWER 17 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN

AN 78278063 EMBASE  
 DN 1978278063  
 TI The **sudden infant death syndrome**:  
 a possible hypersensitivity reaction determined by distribution of IgG in  
 lungs.  
 AU Raven C.; Maverakis N.H.; Eveland W.C.; Ackermann W.W.  
 CS Dept. Epidemiol., Univ. Michigan, Ann Arbor, Mich. 48109, United States  
 SO Journal of Forensic Sciences, (1978) 23/1 (116-128).  
 CODEN: JFSCAS  
 CY United States  
 DT Journal  
 FS 049 Forensic Science Abstracts  
 007 Pediatrics and Pediatric Surgery  
 024 Anesthesiology  
 005 General Pathology and Pathological Anatomy  
 026 Immunology, Serology and Transplantation  
 LA English  
 AB Lung sections and smears from 22 SIDS victims of various ages, exhibiting  
 varying degrees of interstitial pneumonia, gave evidence of bound IgG when  
 examined by the direct fluorescent antibody technique. Appropriate control  
 specimens were negative. All specimens containing IgG failed to exhibit  
 IgM, **IgA**, or IgE. Four specimens containing IgG also contained  
 respiratory syncytial viral antigen. The deposition of IgG and the  
 relationship to the pulmonary lesions in SIDS suggest an immunologic or  
 some phase of a hypersensitivity reaction to be further explored.  
 CT Medical Descriptors:  
 \*hypersensitivity reaction  
 \***sudden infant death syndrome**  
 etiology  
 major clinical study  
 autopsy  
 respiratory system  
 Drug Descriptors:  
 \*immunoglobulin g  
 RN (immunoglobulin g) 97794-27-9

L22 ANSWER 18 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.  
 on STN DUPLICATE 13

AN 77008868 EMBASE  
 DN 1977008868  
 TI **Sudden infant death syndrome** in  
 South Australia. Measurement of serum IgE antibodies to three common  
 allergens.  
 AU Turner K.J.; Baldo B.A.; Carter R.F.; Kerr H.R.  
 CS Clin. Immunol. Unit, Dept. Microbiol., Univ. West. Australia, Perth,  
 Australia  
 SO Medical Journal of Australia, (1975) 2/23 (855-859).  
 CODEN: MJAUAJ  
 DT Journal  
 FS 049 Forensic Science Abstracts  
 007 Pediatrics and Pediatric Surgery  
 017 Public Health, Social Medicine and Epidemiology  
 026 Immunology, Serology and Transplantation  
 LA English  
 AB Radioallergosorbent test (RAST) studies showed that IgE antibodies to  
 Dermatophagoides pteronyssinus (house dust mite), Aspergillus fumigatus  
 and bovine .beta. lactoglobulin were significantly elevated in the sera of  
 infants who died as a result of the sudden death in infancy syndrome  
 (SDIS). No significant differences were found in the levels of total IgE,  
**IgA**, IgG or IgM in the sera of SDIS victims or controls. The  
 possible role of hypersensitivity in the aetiology of SDIS is discussed.  
 CT Medical Descriptors:

\*aspergillus fumigatus  
 \*dermatophagoides pteronyssinus  
 \*hypersensitivity  
 \*immunoglobulin blood level  
 \*infant  
 \*serum  
 \*sudden death  
 autopsy  
 major clinical study  
 etiology  
 diagnosis  
 child  
 fatality  
 Drug Descriptors:  
 \*antibody  
 \*immunoglobulin e

RN (immunoglobulin e) 37341-29-0

L22 ANSWER 19 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN

AN 76206718 EMBASE

DN 1976206718

TI Sudden death in infants.

AU Editorials

CS Canada

SO Canadian Medical Association Journal, (1975) 113/9 (809-812).

CODEN: CMAJAX

DT Journal

FS 005 General Pathology and Pathological Anatomy

007 Pediatrics and Pediatric Surgery

026 Immunology, Serology and Transplantation

LA English

AB In recent mth several theories have been put forward about the cause of death of infants found dead in a crib, and in 2 reports an immunologic reaction is suggested. The fetus carries only such antibodies as it may receive passively, and the infant is born without his own preformed sensitized response. Immunologic competence is therefore acquired somewhat later than birth. The built in period of total breast feeding may span the gap until the baby develops his own competence, by providing a period during which the baby is not absorbing foreign antigens. It seems that the newborn receives a package of antibodies of IgG, IgM and **IgA** classes, augmented by lysozyme, lactoferrin and macrophages present in the milk. The defence is, of course, incomplete, which is perhaps why one has been late in recognizing it. But cot death is more common among bottle fed babies, and there are clues indicating that impairment of the baby's immunologic endowment increases the risk. Death, in this case, may result from an anaphylactic reaction, the antibodies may be of the IgE or short lived IgG class, and the antigen may be from an infecting agent or from food. The low levels of steroid secretion at night may permit transient perforation of membranes or gut absorption or altered sensitivity. Other immunologists, however, are not impressed by an anaphylactic reaction to cow's milk and believe that house dust may be behind the anaphylaxis, especially the mite *Dermatophagoides pteronyssinus*. Some histologic and blood gas analysis data suggested that death might occur from asphyxia due to pulmonary anaphylaxis. It was also found that the prevalence of IgE antibodies to the house dust mite, the mould *Aspergillus fumigatus*, and bovine .beta. lactoglobulin was significantly greater in infants who had died suddenly. The greatest difference was for house mite antibodies, which suggests that hypersensitivity to house dust mite may be a factor in sudden death in infancy (in Western Australia). Some American investigators tried to establish the cardiac response to an auditory stimulus in a study of early learning. The neonatal cardiac response to

such stimulation is an increased heart rate. With repeated stimulation the response decreases, indicating habituation. While 24 infants were being studied, 1 died suddenly at the age of 5 wk and was reported after autopsy to be a victim of the **sudden infant death**

**syndrome**. The findings suggested that the infant at risk for sudden death, although apparently healthy and able to become habituated, had poorly controlled central mechanisms for maintaining stable cardiac function, especially after stimulation. It is suggested that the lability of cardiac response to stimulation may provide an objective measure of dysfunction of the central mechanisms that stabilize cardiac rate and respiratory activity. And so the theories continue, none of them entirely convincing, but all illuminating new facets of this complex problem.

CT Medical Descriptors:

- \*brain
- \*breast feeding
- \*house dust
- \*noise injury
- fatality
- child
- etiology

Drug Descriptors:

- \*immunoglobulin

RN (immunoglobulin) 9007-83-4

L22 ANSWER 20 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.  
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AN 75216858 EMBASE

DN 1975216858

TI IgE antibodies to Dermatophagoides pteronyssinus (housedust mite),  
Aspergillus fumigatus, and .alpha. lactoglobulin in **sudden**  
**infant death syndrome**.

AU Turner K.J.; Baldo B.A.; Hilton J.M.N.

CS Clin. Immunol. Unit, Dept. Microbiol., Univ. West. Australia, Perth,  
Australia

SO British Medical Journal, (1975) 1/5954 (357-360).

CODEN: BMJOAE

DT Journal

FS 026 Immunology, Serology and Transplantation

007 Pediatrics and Pediatric Surgery

013 Dermatology and Venereology

LA English

AB The prevalence of serum IgE antibodies to Dermatophagoides pteronyssinus (house dust mite), Aspergillus fumigatus, and bovine .beta. lactoglobulin was significantly greater in cases of sudden infancy death (SID) than in a control group of infants of the same age range. This difference was most pronounced with D. pteronyssinus antibodies, which suggests that hypersensitivity to housedust mite may be a factor in the etiology of SID in Western Australia. Both the SID and control infants had similar serum concentrations of immunoglobulins G, M, and E but **IgA** levels were significantly higher in the control group.

CT Medical Descriptors:

- \*allergy
- \*antibody production
- \*aspergillus fumigatus
- \*death
- \*dermatophagoides pteronyssinus
- \*hypersensitivity
- \*immediate type hypersensitivity
- \*infancy
- \*skin disease
- major clinical study
- in vitro study

methodology  
etiology  
Drug Descriptors:  
\*antibody  
\*immunoglobulin a  
\*immunoglobulin e  
\*immunoglobulin g  
\*immunoglobulin m  
\*lactoglobulin

RN (immunoglobulin e) 37341-29-0; (immunoglobulin g) 97794-27-9;  
(immunoglobulin m) 9007-85-6

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on STN

AN 75084703 EMBASE

DN 1975084703

TI The immunologic significance of the mammary gland.

AU Beer A.E.; Billingham R.E.; Head J.

CS Dept. Cell Biol., Univ. Texas Southwest. Med. Sch., Dallas, Tex. 75235,  
United States

SO Journal of Investigative Dermatology, (1974) 63/1 (65-74).

CODEN: JIDEAE

DT Journal

FS 013 Dermatology and Venereology

005 General Pathology and Pathological Anatomy

010 Obstetrics and Gynecology

026 Immunology, Serology and Transplantation

LA English

AB The immunologic role of the mammary gland is to transfer ready made antibodies, the 'maternal immunologic endowment', from the maternal serum to the gastrointestinal tract and eventually the blood stream of the immunologically naive infant. In certain species, the mammary gland deputizes for the fetal membranes which are incapable of transmitting antibodies, and in others it continues a process initiated in utero. Antibody secretion by the mammary gland is highly selective. Certain antibodies are preferentially taken up from the serum and concentrated. Absorption from the host's gastrointestinal tract is equally selective and of short duration. In man, gastrointestinal antibody absorption does not occur. Despite this, secretory **IgA** antibodies fulfil an immunoprotective role within the lumen of the gut, acting against a variety of enteric microorganisms. These antibodies are synthesized by plasma cells associated with active mammary tissue. Apart from their beneficial roles, mammary glands may exert an inimical role by virtue of the nature of their exosecretions. In some species they transmit maternal isoantibodies that can cause hemolytic disease of the newborn. They secrete certain proteins, such as casein, which are a potent source of allergic reactions, possibly including the '**sudden infant death syndrome**'. Finally, viable leukocytes are a largely neglected but constant ingredient of colostrum and milk. These can reach the circulation of the recipient and can interact with mononuclear cells of the host; as a result, transplantation immunity, tolerance, and graft versus host disease may develop in some species.

CT Medical Descriptors:

\*allergy

\*breast

\*cell

\*colostrum

\*enterobacteriaceae

\*fetus

\*fetus membrane

\*gastrointestinal tract

- \*graft versus host reaction
- \*hemolytic anemia
- \*immunization
- \*immunology
- \*infancy
- \*lactation
- \*newborn
- \*plasma cell
- \*serum
- \*sudden death
- \*histocompatibility
- review
- Drug Descriptors:
- \*antibody
- \*casein
- \*immunoglobulin a
- \*alloantibody
- \*milk

RN (casein) 9000-71-9; (milk) 8049-98-7

L22 ANSWER 10 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 9

AN 1993:343465 BIOSIS

DN PREV199396040465

TI Mucosal immune response in a case of **sudden infant death syndrome**.

AU Gleeson, Maree (1); Clancy, Robert L.; Cripps, Allan W.

CS (1) Hunter Immunology Unit, Royal Newcastle Hosp., PO Box 664J, Newcastle  
NSW 2300 Australia

SO Pediatric Research, (1993) Vol. 33, No. 6, pp. 554-556.  
ISSN: 0031-3998.

DT Article

LA English

AB A prospective study to define the normal patterns of development of mucosal immunity in 263 children provided a unique opportunity to study the mucosal immune response in an infant who unexpectedly died from **sudden infant death syndrome**. The subject initially had a normal pattern of mucosal immune function, which was perturbed after a transient mild upper respiratory tract infection at 3 1/2 wk of age. After the upper respiratory tract infection, there was an increase in mucosal permeability and the appearance of **IgA** and **IgM** in saliva. The unusual features in this case were the degree and the duration of the increases in salivary **IgA** and **IgM** after resolution of the illness. The marked abnormalities suggested a persistent stimulation of the mucosal immune response. The case provides informative data on potential mechanisms of **sudden infant death syndrome** and supports a role for involvement of upper respiratory tract infection.

CC Biochemical Studies - Proteins, Peptides and Amino Acids \*10064

Biochemical Studies - Carbohydrates \*10068

Pathology, General and Miscellaneous - Necrosis \*12510

Respiratory System - Pathology \*16006

Pediatrics \*25000

Immunology and Immunochemistry - Bacterial, Viral and Fungal \*34504

Medical and Clinical Microbiology - General; Methods and Techniques  
\*36001

BC Hominidae \*86215

IT Major Concepts

Biochemistry and Molecular Biophysics; Immune System (Chemical Coordination and Homeostasis); Infection; Pathology; Pediatrics (Human Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine, Medical Sciences)

IT Industry

cattle industry

IT Miscellaneous Descriptors

IMMUNOGLOBULIN M TITERS; VACCINES

ORGN Super Taxa

Bacteria - General Unspecified: Eubacteria, Bacteria; Bovidae:

Artiodactyla, Mammalia, Vertebrata, Chordata, Animalia;

Enterobacteriaceae: Eubacteria, Bacteria; Hominidae: Primates,

Mammalia, Vertebrata, Chordata, Animalia; Mammalia - Unspecified:

Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name

bacteria (Bacteria - General Unspecified); cow (Bovidae); mammal

(Mammalia - Unspecified); microorganism (Microorganisms - Unspecified);

Escherichia coli (Enterobacteriaceae); Hominidae (Hominidae)

ORGN Organism Superterms

animals; artiodactyls; bacteria; chordates; eubacteria; humans;

mammals; microorganisms; nonhuman mammals; nonhuman vertebrates;

primates; vertebrates

cited in  
australian  
study. *WGC*  
10/14/03

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on STN DUPLICATE 8

AN 95119681 EMBASE  
DN 1995119681  
TI Changes in the concentration and distribution of immunoglobulin-producing cells in SIDS palatine tonsils.  
AU Stoltenberg L.; Vege A.; Saugstad O.D.; Rognum T.O.  
CS Department of Pediatric Research, National Hospital, N-0027 Oslo, Norway  
SO Pediatric Allergy and Immunology, (1995) 6/1 (48-55).  
ISSN: 0905-6157 CODEN: PALUEE  
CY Denmark  
DT Journal; Article  
FS 007 Pediatrics and Pediatric Surgery  
026 Immunology, Serology and Transplantation  
LA English  
SL English  
AB Seventeen **sudden infant death syndrome** (SIDS) cases and 9 controls, were examined immunohistochemically with regard to the presence of **IgA**-, **IgM**-, **IgD**, and **IgG**, as well as for the subtypes **IgG1**-, **IgG2**-, **IgC3**-, and **IgG4**-immunocytes. Differences in compartmentalization were also investigated. Differences were demonstrated between SIDS and controls in total number of **IgG** cells per 0.1 mm<sup>2</sup> tissue area (median: 18.3, range: 12.3-30.2 versus median: 6.3, range: 2.0-14.6) ( $p < 0.01$ ), and for **IgA** immunocytes (median: 3.9, range: 2.4-5.0 versus median: 1.5, range: 1.1-3.7) ( $p < 0.05$ ), while no differences were demonstrated for **IgM** cells (median: 1.8, range: 1.2-3.3 versus median: 1.8, range: 0.7-5.6) or **IgD** cells (median: 1.9, range: 0.8-2.9 versus median: 1.6, range: 0.7-2.4). Differences were demonstrated between SIDS and control **IgG** plasma cells in all the four palatine tonsillar compartments; germinal centre ( $p < 0.01$ ), mantle zone ( $p < 0.05$ ), interfollicular area ( $p < 0.01$ ) and reticular epithelium ( $p < 0.01$ ). Furthermore, the number of **IgA** cells was higher in SIDS vs. controls in both the germinal centre (median: 1.4, range: 0.6-2.1 versus median: 0.6, range: 0.3-1.3) ( $p < 0.05$ ) and in the interfollicular area (median: 2.2, range: 1.1-3.1 versus median: 0.5, range: 0.4-2.0) ( $p < 0.01$ ). For **IgM** immunocytes, differences were demonstrated in the germinal centre (median: 1.0, range: 0.4-1.6 versus median: 0.4, range: 0.3-1.3) ( $p < 0.01$ ) as well as in the germinal centre (median: 0.6, range: 0.5-0.8 versus median: 0.4, range: 0.3-0.7) ( $p < 0.01$ ) and in the interfollicular area (median: 1.2, range: 0.8-1.6 versus median: 0.5, range: 0.5-0.7) ( $p < 0.01$ ) in the **IgD** immunocyte group. The total number of **IgG1**- and **IgG3**-immunocytes were increased in SIDS (median 15.6, range: 5.3-58.9 versus median: 2.5, range: 1.5-8.4) ( $p < 0.01$ ) and (median: 3, 6, range: 0.4-8.6 versus median: 1.1, range: 0.4-1.3) ( $p < 0.01$ ) respectively. Furthermore, significantly increased numbers of these two subclasses, were seen in all the four compartments in the SIDS cases. The palatine tonsillar immune system is stimulated in SIDS. Furthermore, the changes being predominantly in the germinal centres and interfollicular areas, are indicating a recent stimulation, and the **IgG**-subgroup response pattern makes a viral protein antigen the most likely stimulant.

CT Medical Descriptors:  
\*immunoglobulin producing cell  
\*palatine tonsil  
\***sudden infant death syndrome**  
article  
clinical article  
controlled study  
female  
germinal center  
human  
human cell

*cited in  
Australasian  
Study  
LV Cook 10/14/03*

human tissue  
immunohistochemistry  
infant  
lymphocyte  
male  
priority journal

Drug Descriptors:

immunoglobulin a: EC, endogenous compound  
immunoglobulin d: EC, endogenous compound  
immunoglobulin g1: EC, endogenous compound  
immunoglobulin g2: EC, endogenous compound  
immunoglobulin g3: EC, endogenous compound  
immunoglobulin g4: EC, endogenous compound  
immunoglobulin m: EC, endogenous compound  
virus antigen  
virus protein

RN (immunoglobulin m) 9007-85-6

L22 ANSWER 8 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 7

AN 1996:69320 BIOSIS

DN PREV199698641455

TI Pulmonary eosinophilia in **sudden infant death syndrome**.

AU Baxendine, Julia A.; Moore, Isabella E. (1)

CS (1) Dep. Histopathol., Level E, Southampton General Hosp., Southampton SO9  
4XY UK

SO Journal of Pathology, (1995) Vol. 177, No. 4, pp. 415-421.  
ISSN: 0022-3417.

DT Article

LA English

AB A recent immunohistochemical study found increased numbers of eosinophils,  
but no mast cells, in the pulmonary parenchyma of infants who died of  
**sudden infant death syndrome (SIDS)**.

The present study tested the hypothesis that this pulmonary eosinophilia  
could be IgE-mediated. Histomorphometry was used to compare the numbers of  
eosinophils, mast cells, and IgG-, **IgA**-, IgM- and IgE-expressing  
lymphoid cells in the lungs of two groups of infants. Twenty-eight  
subjects aged less than 1 year were selected from postmortem records of  
infant deaths between 1989 and 1992. Fourteen were cases of SIDS and these  
infants were matched for age and gender to 14 controls who died of other  
non-pulmonary conditions. Immunohistochemical stains were used and  
positive cells were counted on six peribronchial and six subpleural  
fields. The numbers of eosinophils in both peribronchial and subpleural  
regions were significantly higher in SIDS compared with controls (P=0.0071  
and P=0.041, respectively). The numbers of **IgA**-expressing  
lymphoid cells were also significantly increased in SIDS cases (P=0.042).  
There were no differences in IgG, IgM or IgE expression or in mast cell  
numbers. These results confirmed that pulmonary eosinophils are increased  
in SIDS, but not through an IgE-mediated pathway.

CC Microscopy Techniques - Histology and Histochemistry \*01056

Cytology and Cytochemistry - Human 02508

Biochemistry - Gases \*10012

Biochemical Studies - General 10060

Biochemical Studies - Proteins, Peptides and Amino Acids 10064

Biochemical Studies - Carbohydrates 10068

Anatomy and Histology, General and Comparative - Microscopic and  
Ultramicroscopic Anatomy \*11108

Pathology, General and Miscellaneous - Necrosis \*12510

Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies \*15004

Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and

Reticuloendothelial System \*15008

Respiratory System - Pathology \*16006

Pediatrics \*25000

Immunology and Immunochemistry - Immunopathology, Tissue Immunology  
\*34508

BC Hominidae \*86215

IT Major Concepts

Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport  
and Circulation); Clinical Immunology (Human Medicine, Medical  
Sciences); Methods and Techniques; Morphology; Pathology; Pediatrics  
(Human Medicine, Medical Sciences); Pulmonary Medicine (Human Medicine,  
Medical Sciences)

IT Miscellaneous Descriptors

AIRWAY OBSTRUCTION; HYPOXIA; IMMUNOGLOBULIN A; IMMUNOGLOBULIN E;  
IMMUNOGLOBULIN G; IMMUNOGLOBULIN M; PEDIATRICS

ORGN Super Taxa

Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name

Hominidae (Hominidae)

*Cited in  
Australian  
study - 10/14/03  
L/Cook*

ORGN Organism Superterms

animals; chordates; humans; mammals; primates; vertebrates